BASELINE LEVELS OF CORAL DISEASE IN THE NORTHWESTERN HAWAIIAN ISLANDS

BY

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ABSTRACT

There has been a worldwide increase in the reports of diseases affecting marine organisms. In the Caribbean, mass mortalities among organisms in reef ecosystems have resulted in major shifts in community structure. However, our ability to fully understand recent disease outbreaks is hampered by the paucity of baseline and epidemiological information on the normal disease levels in the ocean. The Northwestern Hawaiian Islands (NWHI) is considered one of the last relatively pristine coral reef ecosystems remaining in the world. As such, it provides the unique opportunity to document the normal levels of disease in a coral reef system exposed to limited human influence.

In July 2003, baseline surveys were conducted at 73 sites throughout the NWHI to quantify and characterize coral disease. Ten disease states were documented with the most common disease found to be *Porites* trematodiasis. This disease was widespread and is known to exclusively affect *Porites* sp. coral. Numerous other conditions were observed but at much lower levels of occurrence. Numbers of colonies affected by *Porites* trematodiasis were not enumerated but other types of conditions were counted with the average prevalence of disease estimated at 0.5%. Several of the observed disease states were distinct from what has been described from other coral reef systems. Coral genera exhibited differences in types of syndromes and prevalence of disease. Pocilloporids, common corals on the reefs of the NWHI, were comparatively resistant to disease. In contrast, acroporids showed the greatest damage from disease and the highest estimated prevalence of disease.

INTRODUCTION

Coral disease is a rising problem on coral reefs worldwide. The numbers of diseases and coral species affected, as well as the distribution of diseases, have all increased within the last decade (Porter et al., 2001; Green and Bruckner, 2000; Sutherland et al., 2004; Weil, 2004). Recent epizootics of coral disease have resulted in significant losses of coral cover. An outbreak of white band disease in the 1980s killed acroporid corals all over the Caribbean substantially decreasing coral cover (Glatfelter, 1982; Aronson and Precht, 2001), and a recent outbreak of white pox disease in the Florida Keys reduced the cover of *Acropora palmata* by up to 70% (Patterson

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et al., 2002). In the Caribbean, coral disease has been implicated as a major factor contributing to the decline of coral reefs, resulting in apparent ecological phase shifts from coral- to algal-dominated ecosystems (Hughes, 1994; Aronson and Precht, 2001; Porter et al., 2001; Sutherland et al., 2004). What has changed in our oceans to produce this unprecedented increase in disease within the last decade? Increased anthropogenic stress on nearshore environments, overfishing, and environmental conditions associated with global climate change have all been implicated as contributing to increased levels of disease (Harvell et al., 1999; Barber et al., 2001). However, our ability to fully understand recent increases in coral disease is hampered by the paucity of baseline and epidemiological information on the normal disease levels in the ocean (Harvell et al., 1999). It is difficult to understand the underlying mechanisms affecting disease occurrence without knowing normal levels of disease in a healthy ecosystem.

The Hawaiian Archipelago consists of the inhabited Main Hawaiian Islands (MHI) and the more remote Northwestern Hawaiian Islands (NWHI), which span ~1,800 kilometers across more than five degrees of latitude in the northern part of the Archipelago (Fig. 1). The NWHI is a series of islands, banks, shoals, and atolls that have been under federal and state protection since 1909. Their remoteness and protected status has spared the NWHI from much of the degradation experienced by most other coral reef systems. The NWHI is considered to be one of the last relatively pristine, large-scale coral reef ecosystems remaining in the world. As such, a unique opportunity exists here to document normal levels of disease in a coral reef system exposed to only limited human influence. In 2000, the NWHI Ecosystem Reserve was established and a series of multi-agency ship-based expeditions were initiated to assess the biodiversity, status, and management needs of the shallow reefs of the NWHI. In 2002, disease assessment was added to the protocol to characterize and investigate the dynamics of coral disease on these reefs. The purpose of this study was to further characterize and quantify coral disease on the reefs of the NWHI.

METHODS AND MATERIALS

Study Area

The NWHI consists of ten island/banks and atolls which include from southeast to northwest: Nihoa, Necker, French Frigate Shoals, Gardner Pinnacles, Maro Reef, Laysan, Lisianski, Pearl and Hermes, Midway, and Kure (Fig. 1). Nihoa and Necker are small basalt islands, each surrounded by a shallow (<50 m) shelf. French Frigate Shoals is an open atoll with a small basaltic pinnacle in the interior. Gardner Pinnacles consists of three small rocks on an extensive submerged bank. Maro Reef is a complex of shallow reticulated reefs with no associated island. Laysan and Lisianski are low carbonate islands that crest shallow, submerged banks. Northwest of these are three atolls: Pearl and Hermes, Midway, and Kure Atolls (Maragos & Gulko, 2002).

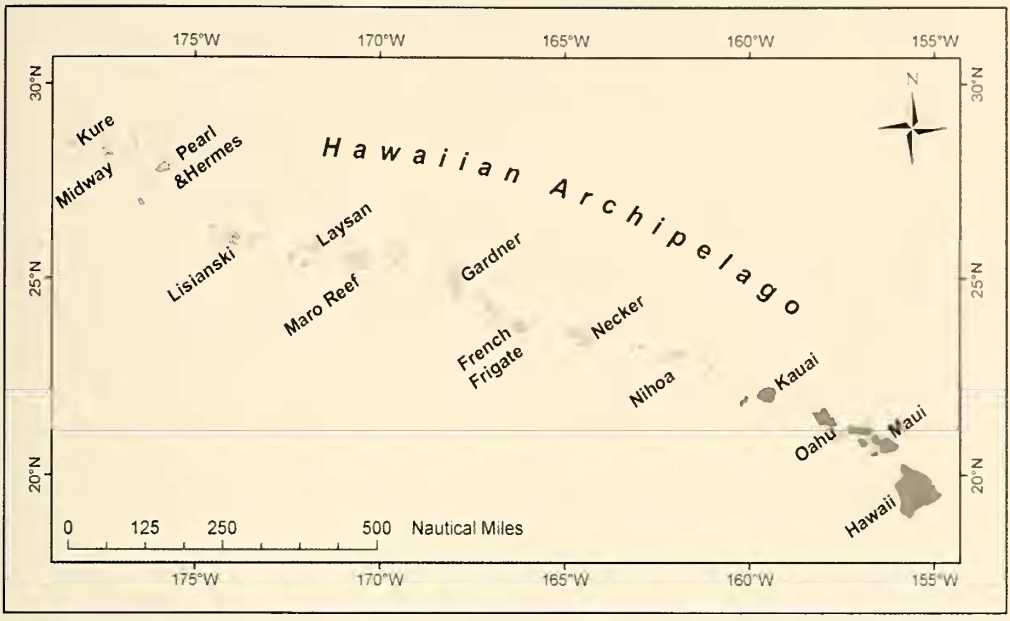

Figure 1. Map of the Hawaiian Archipelago.

Disease Surveys

In July 2003, 73 sites were surveyed for coral disease at nine islands/atolls across the NWHI as part of a long-term monitoring program (Table 1). The 73 sites were selected for long-term monitoring from a pool of 391 sites that had been surveyed during annual research cruises in 2000, 2001, and 2002. Criteria for selection of long-term monitoring sites included representing a range of habitats and biological communities at each location and having a high probability of being accessible to divers on annual research cruises under prevalent sea conditions. At each site, two consecutive 25-m lines, separated by approximately 5 m, were laid out along depth contours. Coral community structure was documented on the first of the two 25-m transect lines by recording coral colonies by size class. All corals, with the colony center within 1 m on either side of the transect line, were enumerated and placed into one of seven size classes: <5, 5-10, 10-20, 20-40, 40-80, 80-160, and >160 cm. These protocols have been used successfully in other studies to document coral community structure within the NWHI (Maragos et al., 2004). Disease assessment was conducted within each 25 x 2m belt transect, as well as, within a wider 25 x 6m belt transect along the 2nd line as time allowed. All coral colonies with disease signs were described, enumerated, and photographed, and samples were collected for follow-up laboratory analyses. Due to time constraints, colonies with the disease *Porites* trematodiasis were not enumerated, but presence or absence of the disease was recorded for each site.

Table 1. Number of sites surveyed for coral disease in the NWHI in July 2003. Sites are categorized by island and reef zone.

Atoll/island	code	zone	# sites surveyed	depth range (ft)	total reef area surveyed for disease (m^2)
Necker	NEC	shelf	3	38-46	375
French Frigate Shoals	FFS	backreef	1	5	100
		forereef	5	10-38	500
		lagoon	6	16-37	1500
Gardner Pinnacle	GAR	shelf	3	40-64	300
Maro Reef	MAR	forereef	6	35-60	600
		lagoon	3	31-52	300
Laysan	LAY	shelf	3	40-48	600
Lisianski	LIS	forereef	3	40-51	600
		lagoon	5	30-56	1000
Pearl & Hermes	PHR	backreef	6	3-22	1200
		forereef	5	39-52	1000
		lagoon	4	26-36	800
Midway Atoll	MID	backreef	4	3-5	800
		forereef	4	38-47	800
		lagoon	3	7-15	600
Kure Atoll	KUR	backreef	3	5-7	600
		forereef	3	36-49	600
		lagoon	3	11-22	600
total			**73**		**12,875**

Statistical Analysis

Time constraints underwater prevented us from enumerating all coral colonies within the wider belt transects surveyed for disease. Therefore, we estimated the total number of colonies surveyed for disease based upon the average number of colonies/m^2 found within the 25x2m belt transect using the following equation:.

number of corals examined for disease per site =
[avg. number of corals per m^2][X total area surveyed for disease (m^2)]

Prevalence of disease was then calculated as follows:

[(number of diseased colonies per site)/(number of colonies examined per site)] 100

To determine overall prevalence of disease for coral genera and disease states, data from all surveys were combined and calculated as follows:

[(number of diseased colonies (all sites combined))/(number of colonies examined (all sites combined))] 100

Overall prevalence was calculated separately for each of the four coral genera (*Acropora, Montipora, Pocillopora, Porites*). For example:

[(number of diseased *Acropora* colonies (all sites combined))/(number of *Acropora* colonies examined (all sites combined))] 100

Overall prevalence was also calculated separately for each disease state with the denominator (# colonies examined) being limited to the specific coral genera affected by that disease state.

Frequency of disease occurrence (FOC) was calculated as:

[(number of sites with disease)/(total number of sites surveyed)] 100

Disease states were categorized by coral genera. FOC of each disease state was calculated as:

[(number of sites having a particular disease state)/(total number of sites containing the affected genera)] 100

For each coral genus, FOC was calculated as:

[(number of sites having disease of each genera)/(number of sites containing that genera of coral)] 100

The data were not normally distributed, even with transformations, therefore non-parametric statistics were applied. Differences in prevalence of coral disease among islands and reef zones were tested using Kruskal-Wallis non-parametric one-way analysis of variance. Differences in overall prevalence of disease among coral genera were tested with a Chi-square test for equality of distributions.

RESULTS

Coral Community Structure

The relative abundance of coral taxa varied by island and by zone within islands (Table 2). In atoll geomorphic systems, backreef zones at the three highest-latitude atolls (Kure, Midway, Pearl and Hermes) are dominated by montiporids and/or pocilloporids, whereas at French Frigate Shoals the backreef is dominated by massive and encrusting *Porites* and other coral (predominantly *Acropora*). At all four atolls, the forereef zone is

Table 2. Summary of colony counts within belt transect surveys conducted at each site. Data reflect the average proportion (%) of colonies within each transect belonging to each of the four dominant genera. Number in parentheses is standard error.

Atoll/island	zone	*Acropora*	*Montipora*	*Pocillopora*	*Porites*
Necker	shelf	0	2.1 (1.1)	40.4 (8.3)	57.5 (9.4)
French Frigate Shoals	backreef	21.6	0	15.7	62.7
	forereef	17.1 (9.8)	6.9 (5.5)	27.1(14.2)	48.9 (9.4)
	lagoon	19.8 (16.3)	3.7 (1.4)	22.3 (11.7)	54.2 (17.3)
Gardner Pinnacle	shelf	0.33 (0.3)	0.35 (0.18	9.1 (2.8)	90.2 (3.2)
Maro Reef	forereef	0.09 (0.09)	25.2 (8.1 0	6.1 (1.7)	68.6 (8.7)
	lagoon	6.4 (3.3)	22.9 (6.7)	23.5 (9.5)	47.1 (17.6)
Laysan	shelf	0	3.2 (1.6)	40.1 (30.0)	56.7 (28.4)
Lisianski	forereef	0	7.0 (3.0)	7.9 (4.1)	84.1 (3.4)
	lagoon	0	33.0 (7.7)	16.3 (6.3)	50.7 (4.5)
Pearl & Hermes	backreef	0	43.7 (19.7)	43.8 (16.5)	12.5 (4.7)
	forereef	0	0	16.0 (11.3)	84.0 (11.3)
	lagoon	0	4.3 (3.0)	27.1 (21.9)	68.6 (21.1)
Midway Atoll	backreef	0	47.8 (27.6)	24.3 (12.0)	27.9 (16.4)
	forereef	0	0	13.9 (8.7)	86.1 (8.7)
	lagoon	0	0	52.9 (27.4)	47.1 (27.4)
Kure Atoll	backreef	0	24.5 (13.2)	42.4 (6.5)	33.1 (18.5)
	forereef	0	0	48.6 (21.6)	51.4 (21.6)
	lagoon	0	0	61.1 (28.7)	38.9 (28.7)

co-dominated by pocilloporids and by massive and encrusting *Porites*. In the lagoon zone, branching *Porites compressa* dominates the coral fauna at Kure and at Pearl and Hermes, whereas massive and encrusting *Porites* along with *Porites compressa* co-dominate the lagoon zone at French Frigate Shoals. Shelf zones surrounding Necker, Gardner Pinnacle, and Laysan are sparsely populated by massive and encrusting *Porites* and by pocilloporids.

Overall Occurrence of Coral Disease

Ten different disease states were documented from the four major coral genera found in the NWHI (Table 3). Coral disease was found at 68.5% of the sites surveyed, but prevalence of disease was low, with an average of 0.5% of the colonies having signs of disease (range=0 - 7.09%). FOC of disease varied among the islands with Laysan and Lisianski having the highest (FOC=100%) and Midway having the lowest (FOC=27.3%)(Table 4).

Prevalence of disease also differed among islands with FFS and Midway having the highest prevalence of disease (Fig. 2). However, intra-island variability was also high, therefore between-island comparisons were not statistically significant (Kruskal-Wallis, X^2=13.2, *df*=8, P=0.1059). Disease prevalence varied among reef zones (Table

Table 3. Description of 10 coral diseases found on the reefs of the NWHI in July 2003. Frequency of occurrence = (# of sites with presence of the disease/# of sites containing affected genera) X 100.

genera	disease	characteristics	distribution	freq of occurrence (%)	host species
Porites	*Porites trematodiasis (TRM)*	3-5mm diameter, pink to pale, swollen nodules on coral colony. Nodules can be clustered or widely distributed on colony.	all islands	69.8	*P. lobata, P. compressa, P. evermanni*
	Porites tissue loss syndrome (TLS)	Irregular patches of tissue loss. Patches usually bordered by a narrow, bleached, pink or mucous band. Older exposed skeleton is algae-colonized.	FFS, MAR, PHR, MID, KUR	15.9	*P. lobata, P. evermanni*
	Porites discolored tissue thinning syndrome (DTTS)	Areas of tissue thinning and discoloration that are poorly defined from surrounding healthy tissue. Polyps are reduced or absent.	FFS, MAR, LAY, LIS, PHR, KUR	22.2	*P. lobata*
	Porites brown necrotizing disease (BND)	Diffuse, well-defined, areas of dark brown discoloration characterized by a gelatinous texture and loss of recognizable polyp structure.	PHR	3.2	*P. lobata*
Montipora	*Montipora tissue loss syndrome (TLS)*	Well-defined areas of tissue loss revealing intact white skeleton. Border between healthy and diseased tissue usually with band of mucous, bleached tissue, or thin (1 polyp deep) layer of white necrotic tissue. Older exposed skeleton is algae-colonized.	MAR, LAY, MID	21.1	*M. patula, M. capitata, M. turgescens, M. verrilli*
	Montipora patchy tissue loss (PTL)	Multiple, well-defined circular areas of tissue loss revealing intact white skeleton. Can have residual necrotic tissue in center. Lesions usually ~ 5mm in diameter but can coalesce to form larger areas.	MAR	2.6	*M. patula*
	Montipora growth anomaly (GA)	Well-defined areas of excess skeletal growth. Tissue overlying growth anomaly usually paler with calices reduced to absent.	PHR	2.6	*M. capitata*

Table 3. Continued.

Acropora	*Acropora white syndrome (WS)*	Well-defined areas of tissue loss revealing intact white skeleton. Pattern of tissue loss can be patchy or can appear as a linear pie wedged area of tissue loss extending from the center of the table coral to the outer edge. Older exposed skeleton is algae-colonized.	FFS	9.1	*A. cytherea*
	Acropora growth anomaly (GA)	Well-defined areas of excess skeletal growth. Anomalies can range in size from < 1cm to >35cm in diameter. Two types have been described (Work and Rameyer, 2002). One type is compact with reduced calyx structure and the other type has elongated, malformed calices.	FFS	18.2	*A. cytherea*
Pocillopora	white band disease (WBD)	Narrow, linear band of tissue loss revealing bare skeleton.	PHR	1.4	*P. meandrina*

Table 4. Frequency of occurrence of coral disease within islands/atolls of the NWHI. Frequency of occurrence = (# sites with diseased coral/# sites surveyed) x 100.

island/atoll	**# sites surveyed**	**# sites w/ diseased coral**	**freq of occurrence (%)**
Necker	3	1	33.3
French Frigate Shoals	12	8	66.7
Gardner Pinnacle	3	2	66.7
Maro	9	8	88.9
Laysan	3	3	100
Lisianski	8	8	100
Pearl & Hermes	15	10	66.7
Midway	11	3	27.3
Kure	9	7	77.8
total	73	50	68.5

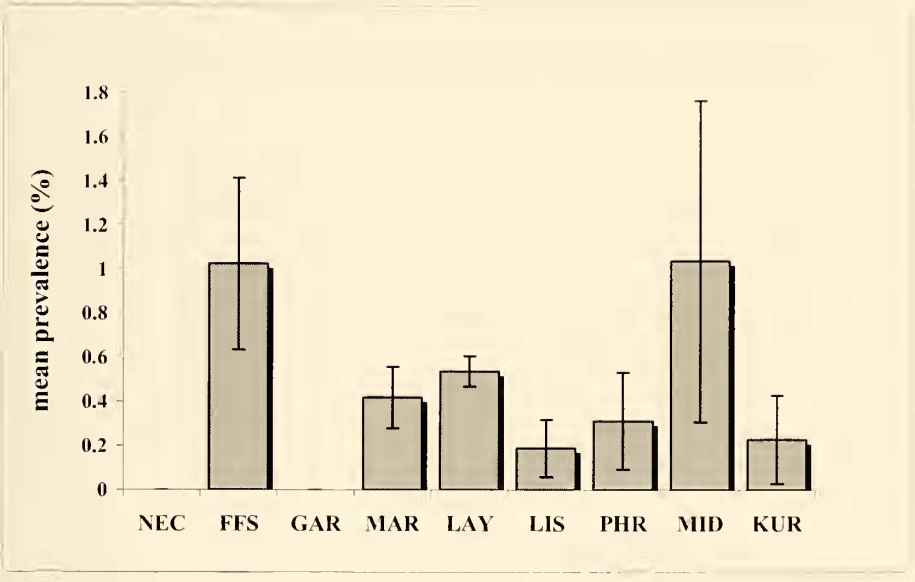

Figure 2. Mean prevalence (+SE) of coral disease at sites across the NWHI. Seventy-three sites were surveyed in July 2003. Prevalence = (# diseased corals/total # corals) X 100. NEC=Necker; FFS=French Frigate Shoals; GAR=Gardner; MAR=Maro; L=Laysan; LIS=Lisianski; PHR=Pearl and Hermes; MID=Midway; KUR=Kure.;

5), but again variability was high, and among-zone comparisons were not statistically significant (Kruskal-Wallis, X^2=4.44, *df*=3, P=0.2176). Disease prevalence varied among coral genera with *Acropora* having the highest prevalence of disease and *Pocillopora* having the lowest (X^2=125.1, *df*=3, P<0.0001; Fig. 3).

Distribution, Frequency of Occurrence, and Prevalence of Each Disease State

Distribution of the different coral diseases varied widely. Some diseases, such as *Porites* trematodiasis, were widespread (occurring at all islands surveyed), whereas others, such as *Pocillopora* white band disease only occurred at a single site (Table 3). The frequency of occurrence of the different diseases followed a similar pattern with some of the most widely distributed diseases such as *Porites* trematodiasis also being the most frequently encountered (69.8% of the sites containing *Porites*). Other common diseases included *Porites* discolored tissue thinning syndrome (FOC=22.2%) and *Montipora* tissue loss syndrome (FOC=21.1%). Other diseases were encountered less frequently during surveys (Table 3).

Prevalence of the different diseases varied with *Acropora* growth anomalies having the highest prevalence (1.85%) and *Porites* brown necrotizing disease having the lowest (0.012%) (Fig. 4).

Table 5. Average prevalence of disease within the different reef zones in the NWHI. Surveys were conducted in July 2003. Prevalence = (# diseased corals/total # corals) x 100. Number in parentheses is standard error.

reef zone	Atoll/island	# sites surveyed	avg. prevalence (%)
Backreef			
	Kure	3	0.62 (0.62)
	Midway	4	2.9 (1.8)
	Pearl & Hermes	6	0.096 (0.06)
	FFS	1	0
	total	**14**	**0.99 (0.57)**
Forereef			
	Kure	3	0.074 (0.037)
	Midway	4	0
	Pearl & Hermes	5	0.83 (0.64)
	FFS	5	0.97 (0.49)
	Maro	6	0.44 (0.17)
	Lisianski	3	0.502 (0.27)
	total	**26**	**0.51 (0.16)**
Lagoon			
	KUR	3	0
	MID	3	0
	PHR	4	0
	LIS	5	0
	MAR	3	0.38 (0.28)
	FFS	6	1.2 (0.69)
	total	**24**	**0.36 (0.20)**
Shelf			
	LAY	3	0.54 (0.069)
	GAR	3	0
	NEC	3	0
	total	**9**	**0.18 (0.09)**

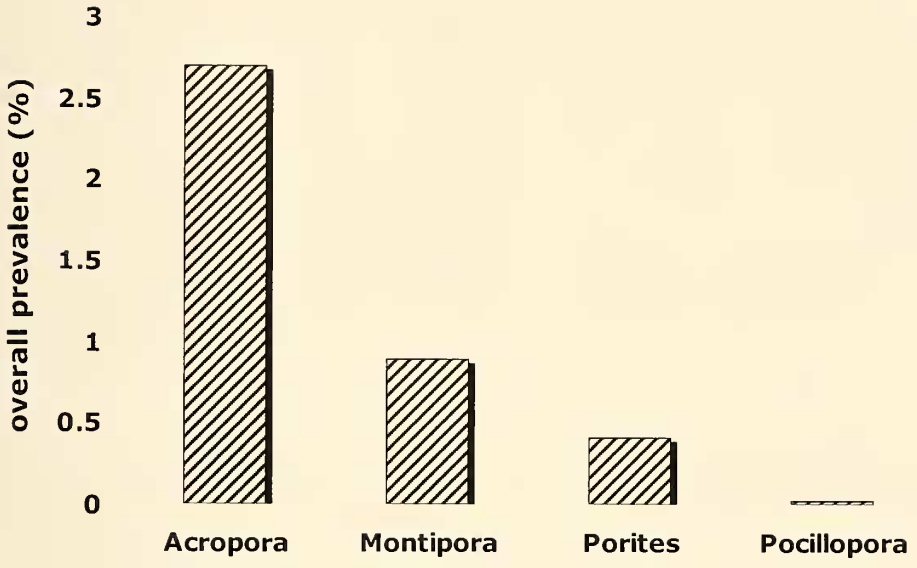

Figure 3. Overall prevalence of disease in the four major coral genera in the NWHI. Seventy-three sites were surveyed in July 2003. Prevalence (all surveys combined) is calculated as the number of diseased colonies per genera/total number of colonies per genera X 100.

DISCUSSION

Approximately 0.5% of the corals examined were found to have signs of disease on the pristine reefs of the NWHI. These findings are important as they allow the level of coral disease in a healthy coral-reef ecosystem to be compared with coral reefs impacted by humans, both within the Hawaiian Archipelago and in other regions of the world. Disease levels found in the NWHI were much lower than what has been reported for other reefs, both in the Indo-Pacific and the Caribbean. Willis et al. (2004) surveyed eight sites along the Great Barrier Reef (GBR) and found the prevalence of disease in hard corals to range from 7.2-10.7%. Raymundo et al. (in press) surveyed eight sites in the Philippines and reported an overall prevalence of disease of 14.2%. In the Caribbean, Weil (2004) reported an average prevalence of 5.28% for surveys conducted at 28 sites from nine regions across the wider Caribbean. Santavy et al. (2001) assessed coral disease at 32 stations throughout the Florida Keys and found disease prevalence to range from 1.0% to 28.2% (avg. 9.6%).

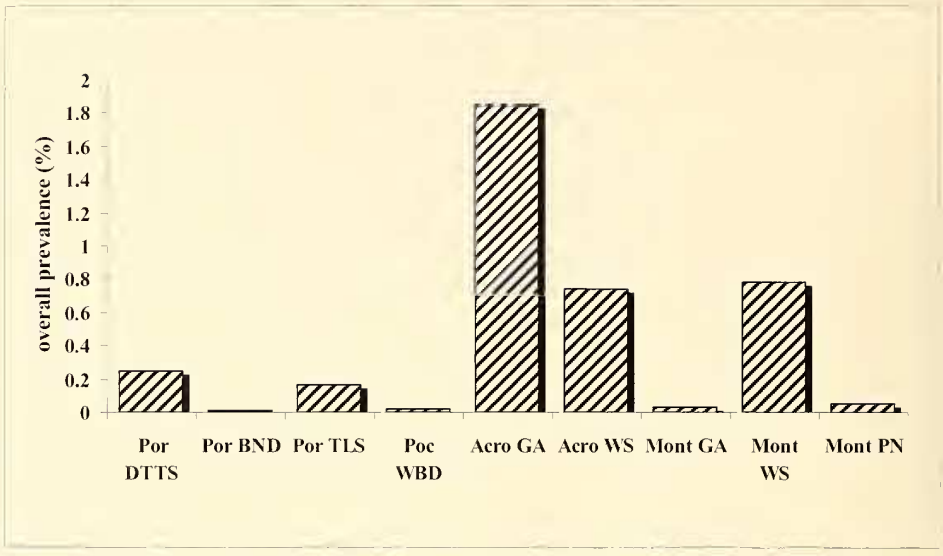

Figure 4. Overall prevalence of each disease state in the NWHI (73 sites surveyed in July 2003). Prevalence (all surveys combined) per disease state is calculated as the number of diseased colonies/ total number of colonies of the affected genera X 100. Por DTTS=*Porites* discolored tissue thinning syndrome; Por BND=*Porites* brown necrotizing disease; Por TLS=*Porites* tissue loss syndrome; Poc WBD= *Pocillopora* white band disease; Acro GA=*Acropora* growth anomaly; Acro WS=*Acropora* white syndrome; Mont GA=*Montipora* growth anomaly; Mont TLS=*Montipora* tissue loss syndrome; Mont PTL=*Montipora* patchy tissue loss.

Ten coral disease states are described from the four major coral genera on the reefs of the NWHI. Four diseases were found to affect *Porites*, three affected *Montipora*, two affected *Acropora,* and one affected *Pocillopora.* In other areas of the Indo-Pacific, similar numbers of diseases are being reported. Six disease states were described from the Philippines (Raymundo et al., in press), and eight categories of disease have been described from the Great Barrier Reef (GBR) (Willis et al., 2004). However, on the GBR, all corals with tissue loss were classified as white syndrome regardless of coral genera or distinctive patterns of tissue loss, and thus eight categories represent a conservative number of disease states. In contrast, 22 diseases have been recorded from the Caribbean (Green and Bruckner, 2000; Sutherland et al., 2004; Weil, 2004). However, research on coral disease in the Caribbean has been ongoing for the past 30 years whereas disease research in the Indo-Pacific only recently has been initiated. For example, this study is the first quantitative disease survey ever conducted in the NWHI. The numbers of diseases described from the Indo-Pacific will no doubt increase as more areas are explored.

Disease signs similar to 7 of the 10 reported disease states within the NWHI have also been reported from other areas of the Indo-Pacific. *Porites* trematodiasis has a widespread distribution across the Indo-Pacific having been reported from Australia (Willis et al., 2004), Main Hawaiian Islands (Aeby, 1998a), and Okinawa (Yamashiro, 2004). *Montipora* tissue loss syndrome and *Porites* tissue loss syndrome are reported

from Australia (Willis et al., 2004) and the Philippines (Raymundo et al., in press). *Acropora* white syndrome and *Pocillopora* white band disease are reported from Australia (Willis et al., 2004). Growth anomalies in both *Acropora* and *Montipora* have been recorded from Australia (Willis et al., 2004), Johnston Atoll (Work et al., 2001), American Samoa (Work and Rameyer, 2002) and Okinawa (Yamashiro et al., 2000, 2001; Yamashiro, 2004). *Pocillopora* white band disease is the only disease found in the NWHI that is similar to what has been described from the Caribbean. It must be noted that there are regional differences in names assigned each set of field disease signs. For example, swollen pink spots on *Porites* are called *Porites* trematodiasis in Hawaii, pink spot in Australia, and *Porites* pink block disease in Okinawa. It is hoped that through the efforts of the Coral Disease and Health Consortium (CDHC) (www.coral.noaa.gov/coral_disease/edhe.shtml) that this nomenclature problem will eventually be resolved. It should also be noted that any similarities in field signs of disease between regions does not necessarily imply the diseases have the same etiology.

Three of the disease states found in the NWHI have not yet been described from elsewhere in the world. They include *Montipora* patchy tissue loss (although this may have been reported as white syndrome in Australia), *Porites* tissue thinning syndrome, and *Porites* brown necrotizing disease. Whether these diseases are specific to Hawaii or not remains to be seen, as studies elsewhere in the Indo-Pacific are still very limited. Much more work is needed to document the occurrence, distribution, etiology, and transmission of diseases across the Indo-Pacific.

The distribution and frequency of occurrence of the different coral diseases varied widely within the nine islands/atolls of the NWHI. Some diseases were both widespread and encountered frequently while other disease states were quite rare. One factor affecting disease occurrence is the distribution of their host populations. Acroporids are limited to five islands/atolls within the NWHI (Necker, French Frigate Shoals, Gardner Pinnacle, Maro, Laysan). The abundance and diversity of *Acropora* is highest on the reefs at French Frigate Shoals (Grigg, 1981; Grigg et al., 1981; Maragos et al., 2004) which is also the only place acroporid disease was found. In contrast, *Porites* is the dominant coral on the reefs of the NWHI comprising 63.5% of the overall coral community within our transects and found at all islands. Accordingly, poritid diseases had both a wider distribution and higher frequency of occurrence than did acroporid diseases. In fact, the most common and widespread disease was *Porites* trematodiasis. In other reef systems where *Porites* is less common, *Porites* trematodiasis is also less common (Willis et al., 2004). However, host distribution is not the only factor controlling disease occurrence, as some poritid diseases, such as *Porites* brown necrotizing disease, were found to be quite rare (FOC=2.7%).

Other factors associated with a pathogen's life history also are important in determining its relative success. Where its coral host is abundant, *Porites* trematodiasis is quite successful, and this can be explained by the attributes of its life history. *Porites* trematodiasis is caused by the encystment of the larval stage of a digenetic trematode in the coral host (Cheng and Wong, 1974; Aeby, 1998a). Completion of the parasite's life cycle occurs when coral-feeding fish ingest the infected polyp, with the adult worm subsequently residing in the guts of fish (Aeby, 1998b). The encysted stage of

the parasite within the coral host can last for several months before senescence of the parasite (Aeby, 1998a). The pink, swollen appearance of the infected polyp attracts fish that preferentially feed on the infected polyps (Aeby, 1992 and 2002). Both of these attributes, the ability to stay viable for long periods of time awaiting transmission and the altered appearance of the coral host, result in an increased probability of successful transmission into the final fish host. Fecal release of the parasite's eggs into the environment from the fish host facilitates transmission of this disease across the reef. Little is known about the etiology or ecology of other diseases, but when more information is available, a clearer picture of the proximate factors controlling disease occurrence should emerge.

Patterns in disease prevalence among the coral genera suggest *Acropora* is the most susceptible to disease and *Pocillopora* is the most resistant. *Acropora* comprised only 2.2% of the overall coral community along our transects. Yet, acroporids showed the highest overall prevalence of disease with *Acropora* growth anomalies having the highest prevalence of all described diseases. *Acropora* white syndrome also resulted in the greatest amount of damage of any of the diseases. An outbreak of *Acropora* white syndrome at one site at FFS resulted in massive tissue loss from numerous large table corals (*A. cytherea*). Tissue loss was visually estimated as ranging from 10-60% of the affected colonies (Aeby, in press). Acroporids have also been greatly affected by disease in Australia (Willis et al., 2004) and have been decimated by disease in the Caribbean (Green and Bruckner, 2000; Porter et al., 2001; Patterson et al., 2004; Weil, 2004). Acroporids were one of the major frame-building corals in the Florida Keys, but losses of acroporids are now averaging 87% or greater (Miller et al., 2002; Patterson et al., 2002; Sutherland et al., 2004).

Hawaii differs from other regions in the exceptionally low occurrence of disease in pocilloporids. In Australia, Willis et al. (2004) found pocilloporids to have the highest prevalence of disease among all coral families surveyed despite pocilloporids having the lowest coral cover. In contrast, pocilloporids are a common coral in the NWHI (21.1% of the overall coral community along our transects) yet seldom showed signs of disease. In fact, an estimated 6,081 pocilloporid colonies were examined during our surveys with only a single colony exhibiting any signs of disease. This suggests that pathogens do not necessarily affect the most common or abundant corals. It also raises the question as to why pocilloporids within the NWHI are so disease free. It could be that the pocilloporids within the NWHI possess inherent mechanisms of defense against disease not found in corals from other regions. Alternatively, since the studies in Australia were conducted on more impacted reefs than found in the NWHI, it may suggest that pocilloporids could be sensitive to certain stressors which makes them more susceptible to disease. Future surveys planned for the impacted reefs of the inhabited Main Hawaiian Islands may shed light on this question.

The distribution and levels of overall disease differed among the nine islands/ atolls surveyed. The occurrence of disease would depend on a number of factors, such as host density, host susceptibility, environmental conditions, or mode of transmission, among others. The NWHI encompasses a variety of reef habitats including shallow backreefs, deeper forereefs, and protected lagoonal reefs. Each reef zone has a unique

set of environmental conditions that influence both coral community structure and overall coral cover. These differences in coral community among reef zones could explain variability in coral disease found among islands. For example, Nihoa, Necker, and Gardner are all high islands surrounded by deeper, forereef environments. These islands experience high wave energy in the winter months, therefore their coral communities are low density encrusting *Porites lobata* and scattered colonies of *Pocillopora meandrina* (Maragos et al, 2004). Accordingly, these sites have few disease states and a low overall prevalence of disease. In contrast, the atoll environments encompass forereef, backreef, and lagoonal reef environments. The number of coral species and colony densities are greater, as well as the number of disease states and prevalence of disease.

Differences in coral community also varied within reef zones and thus affected the level of disease found within zones. For example, at Midway Atoll some backreefs are dominated by montiporids that are more susceptible to disease as compared to other backreefs dominated by the more disease resistant pocilloporids. It is the taxon of corals found on a reef, regardless of which island or reef zone, that primarily affects the types and levels of disease that will occur.

Levels of disease also were also affected by disease outbreaks at two of the atolls (French Frigate Shoals and Midway). At French Frigate Shoals, there was an outbreak of white syndrome on acroporids at one site (prevalence =4.1%), and at Midway there was a high prevalence of *Montipora* tissue loss syndrome at one site (prevalence=7.1%). Interestingly, the montiporids at the site at Midway had experienced a severe bleaching event the year prior (2002) (Aeby et al., 2003; Kenyon et al., in press). The relationship between bleaching stress and disease susceptibility is one that should be investigated more thoroughly especially in light of the predicted increases in bleaching events associated with global climate change (Hughes et al., 2003)

With increased human populations, the scale of human impacts on reefs has grown exponentially. Compounding these anthropogenic stressors are the impacts of global climate change, predicted to result in more frequent bleaching episodes and higher levels of disease (Hughes et al., 2003). Although disease is a natural component of all ecosystems, levels of disease that are higher than expected or changes in levels of disease through time could be indicative of underlying problems. This study of coral disease on the pristine reefs of the NWHI provides an estimate of the normal levels of disease expected on a healthy reef with minimal impact from anthropogenic stress. In this study, colonies with *Porites* trematodiasis were not enumerated; therefore, the prevalence of disease reported here is quite conservative. However, this study combined with further work in the NWHI, which includes enumeration of *Porites* trematodiasis, will serve as an important baseline for comparison with other regions and for monitoring disease levels through time. From these studies, a clearer picture should emerge of the underlying mechanisms that may be influencing the levels of disease found on coral-reef ecosystems throughout the world.

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